FILE 'CAPLUS, MEDLINE, BIOSIS, CA, EMBASE, USPATFULL' ENTERED AT 14:15:52 ON 17 DEC 2003

L1	136	S MEGSIN OR TP55 OR SERPINB7 OR (SERPIN (W) B7) OR SPB7
L2	2931099	S ANTIBOD? OR IMMUNOGLOB?
L3	62	S L1 AND L2
L4	37	DUPLICATE REM L3 (25 DUPLICATES REMOVED)
L5	27	S L1 (S) L2
L6	12	DUPLICATE REM L5 (15 DUPLICATES REMOVED)

L4 ANSWER 35 OF 37 MEDLINE on STN DUPLICATE 14

TI Tissue distribution and biochemical and functional properties of **Tp55** (CD27), a novel T cell differentiation antigen.

AB Two monoclonal **antibodies** (CLB-CD 27/1 and CLB-CD 27/2) were raised against a novel determinant on human T lymphocytes. One of these **antibodies**, CLB-CD 27/1 (clone 9F4), was grouped by the Third International Workshop and Conference on Human Leucocyte Differentiation Antigens together with three other monoclonal **antibodies** (VIT 14, OKT 18A, and S152) in the new cluster CD27. In this paper we show that **antibodies** belonging to this cluster recognize an antigen present on a large subset of peripheral T lymphocytes and most medullary thymocytes. At least two different nonoverlapping epitopes were identified with directly labeled monoclonal **antibodies**. Immunoprecipitation studies indicate that the target antigen of CD27 **antibodies** is a polypeptide of 55 kDa, which appears in the form of a disulfide-linked homodimer on the T lymphocyte membrane (**Tp55**). Stimulation of T cells via the T3/T cell antigen-receptor complex, with either phytohemagglutinin or CD3 monoclonal **antibodies**, resulted in a fivefold increase in the membrane expression of **Tp55**, whereas activation by phorbol myristate acetate caused a marked down-regulation. Moreover, an additional molecule of 32 kDa was precipitated from the membrane of activated but not of resting T cells. Addition of CD27 **antibodies** to cultures stimulated with either phytohemagglutinin or CD3 monoclonal **antibody** led to enhanced proliferation, whereas no effect was observed in phorbol myristate acetate or interleukin 2-stimulated cultures. The possible role of the **Tp55** antigen in T cell activation is discussed.

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L1 136 S MEGSIN OR TP55 OR SERPINB7 OR (SERPIN (W) B7) OR SPB7
L2 2931099 S ANTIBOD? OR IMMUNOGLOB?
L3 62 S L1 AND L2
L4 37 DUPLICATE REM L3 (25 DUPLICATES REMOVED)
L5 27 S L1 (S) L2
L6 12 DUPLICATE REM L5 (15 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 14:22:16 ON 17 DEC 2003

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, USPATFULL' ENTERED AT 14:26:08 ON 17 DEC 2003

FILE 'STNGUIDE' ENTERED AT 14:26:11 ON 17 DEC 2003

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, USPATFULL' ENTERED AT 14:32:26 ON 17 DEC 2003

FILE 'STNGUIDE' ENTERED AT 14:32:27 ON 17 DEC 2003

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, USPATFULL' ENTERED AT 14:33:04 ON 17 DEC 2003

FILE 'STNGUIDE' ENTERED AT 14:33:04 ON 17 DEC 2003

FILE 'CAPLUS, MEDLINE, BIOSIS, CA, EMBASE, USPATFULL' ENTERED AT 14:33:56 ON 17 DEC 2003

L7 7 S L1 AND IMMUNOASSAY
L8 4 DUPLICATE REM L7 (3 DUPLICATES REMOVED)

L6 ANSWER 7 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Association of a uteroglobin polymorphism with rate of progression patients with IgA nephropathy.

AB Uteroglobin gene - disrupted mice develop a nephritis very similar to **immunoglobulin** A (IgA) nephropathy. **Megsin** codes for a protein overexpressed in mesangium in patients with IgA nephropathy. Both are candidate genes that might have variants associated with an accelerated progression in patients with IgA nephropathy. We performed an association study of patients with IgA nephropathy and matching control subjects to test whether the G38A polymorphism in the uteroglobin gene, the C2093T polymorphism in the **megsin** gene, or the angiotensin-converting enzyme (ACE) insertion/deletion polymorphism is associated with IgA nephropathy or rate of disease progression in patients with IgA nephropathy. Of 110 patients with IgA nephropathy, 87 patients were followed up for at least 3 years for the progression study. We also studied 104 healthy volunteers. The uteroglobin, **megsin**, and ACE polymorphisms were not distributed differently in the 110 patients with IgA nephropathy compared with healthy controls; Hardy-Weinberg equilibrium criteria were fulfilled. The GG genotype of the G38A uteroglobin polymorphism was more common in patients with progression (odds ratio [OR], 3.5; $P < 0.006$) than the AG+AA genotypes. The G allele was also more common (OR, 2.6; $P < 0.009$) in patients with versus without progression. The 1/serum creatinine over time plot (in deciliters per milligram per day) was sevenfold steeper in GG patients than the other two genotypes ($P = 0.08$). No significant associations with disease progression were found for the other gene polymorphisms, and a multivariate analysis showed no interactions. We suggest the hypothesis that the uteroglobin gene contains variant(s) with a bearing on progression rate in patients with IgA nephropathy. (C) 2000 by the National Kidney Foundation, Inc.

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